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ORIGINAL PAPER

Assessment of myocardial changes in athletes with native T1 mapping and cardiac functional evaluation using 3 T MRI

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Abstract Intensive physical exercise leads to increases in left ventricular muscle mass and wall thickness. Cardiac magnetic resonance imaging allows the assessment of functional and morphological changes in an athlete's heart. In addition, a native T1 mapping technique has been suggested as a non-contrast method to detect myocardial fibrosis. The aim of this study was to show the correlation between athletes' cardiac modifications and myocardial fibrosis with a native T1 mapping technique. A total of 41 healthy non-athletic control subjects and 46 athletes underwent CMR imaging. After the functional and morphological assessments, native T1 mapping was performed in all subjects using 3.0 T magnetic resonance imaging. Most of the CMR findings were significantly higher in athletes who had ≥5 years of sports activity when compared with non-athletic controls and athletes who had <5 years of sports activity. Significantly higher results were shown in native T1 values in athletes who had <5 years of sports activity, but there were no significant differences in the left ventricular end-diastolic volume, left ventricular end-diastolic mass, or interventricular septal wall thickness between non-athletic controls and athletes who had <5 years of sports activity. The native T1 mapping technique has the potential to discriminate myocardial fibrotic changes in athletes when compared to a normal myocardium. The T1 mapping method might be a feasible technique to evaluate athletes because it does not involve contrast, is non-invasive and allows for easy evaluation of myocardial remodeling.

Keywords Athlete's heart · Cardiac magnetic resonance imaging · Native T1 mapping technique · 3 T MRI

Introduction

Intensive continual exercise leads to cardiac modifications and remodeling, which are known as 'athlete's heart'. Left ventricular hypertrophy (LVH), left ventricular (LV) and right ventricular (RV) enlargement, and bradycardia are some of the cardiac changes observed in athletes [1, 2]. A study about sudden cardiac death (SCD) in athletes showed that 14 % of cases involved idiopathic myocardial fibrosis with or without LVH [3]. A study using cardiac magnetic resonance (CMR) imaging found that 12 % of 102 marathon runners had myocardial scarring. The prevalence in this group was three times greater than that observed in an age-matched control group [4]. Multiple cardiac diseases can result in myocardial fibrosis, which can progress to heart failure and SCD. However, the significance of exercise-induced fibrosis is not well known in terms of arrhythmogenic focus. Some authors have asserted that the intensity of endurance exercise may have adverse cardiovascular effects that outweigh the benefits [5–7]. At the same time, although athletic heart syndrome is considered a physiologic response to exercise in general, the efficacy

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of endurance training on cardiac remodeling in young athletes requires more research [8, 9].

CMR imaging is increasingly used to evaluate LV wall thickness and to perform cardiac functional assessments to identify cardiac disease in athletes. However, quantitative ventricular parameters alone are not sufficient to evaluate the risk of SCD because they do not detect myocardial fibrosis [3]. Although late gadolinium enhancement (LGE) makes it possible to identify localized fibrosis, it usually overlooks diffuse myocardial fibrosis and allows only a qualitative assessment [10]. Recently, the native T1 mapping technique using CMR imaging has shown promise in evaluating myocardial fibrosis. Some studies have shown that diffuse myocardial fibrosis evaluated by histology correlates to native T1 values and have demonstrated that T1 mapping can be comparable to LGE imaging for detecting myocardial scar extensions [11, 12].

The aim of this study was to evaluate the correlation between athletes' cardiac modifications and myocardial fibrosis by 3.0 T CMR imaging using the native T1 mapping technique. We hypothesized that native T1 values increase in athletes' hearts compared with normal volunteers. We also hypothesized that T1 values could be used to differentiate between athletes with and without LVH, which could guide future investigations on the etiological identification of SCD and proper preventive and screening protocols.

Methods

This prospective, observer-blinded study was performed in a single tertiary center, and the study was approved by the local medical ethics committee. All participants provided written informed consent.

Study population

Between March and November 2015, 41 healthy non-athletic controls (group 1) and 46 athletes (group 2) underwent CMR imaging at our radiology department. Inclusion criteria were a minimum of 6 h of intense exercise per week for the athletes and a maximum of 3 h of moderate exercise per week for the control group. Cardiovascular adaptations to exercise vary depending on the type of athletic training, which influences the degree of hypertrophy. To standardize cardiac remodeling, the athletic group consisted of moderate- to high-dynamic and low-static athletes (long distance running, football, volleyball, basketball, tennis), based on the classification of sports criteria [13], with no history of cardiac events. Exclusion criteria for both groups were known LVH, cardiomyopathy, aortic stenosis, heart valve disease, hypertension, contraindications for CMR or a familial history of cardiovascular disease. Before CMR imaging, the arterial blood pressure of all subjects was measured, and blood pressure values >140 mmHg systolic and >90 mmHg diastolic were deemed hypertensive [14]. In addition, all participants avoided training for 3 days before CMR imaging to prevent false increases in T1 values in response to acute changes in the myocardium.

Cardiac magnetic resonance imaging

All athletes and healthy non-athletic controls underwent CMR imaging using a 3.0 T clinical scanner (Magnetom Skyra, Version E11, Siemens Healthcare, Erlangen, Germany) with an 18-channel cardiac coil and electrocardiography gating. After standardized CMR planning [15], standard steady-state free-precession cine images in a short axis covering the LV and heart chambers 2, 3, and 4 were acquired with the following image parameters: TR, 41 ms; TE, 1.2 ms; flip angle, 47°. In addition, native T1 mapping was performed in a single mid-ventricular short-axis slice using the modified look-locker inversion recovery (MOLLI) sequence (TR, 280.6 ms; TE, 1.12 ms; flip angle, 35°) in all subjects. The sequence was derived from the pixelwise T1 calculation from inversion time (TI)-dependent signal intensities in motion-corrected MOLLI images. T2 mapping was performed to exclude the edematous myocardial zones related to such cardiac pathologies as myocarditis and acute myocardial infarction [16, 17]. T2 mapping data were acquired in the same mid-ventricular short-axis plane used in T1 mapping, and the T2-weighted SSFP technique (TR, 207.4 ms; TE, 1.32 ms; flip angle, 12°) was used in all subjects. The acquired images were stored in DICOM format.

Image analysis

All CMR image analyses were performed on a Syngo Via (Software Version VA30A, Siemens AG, Germany) workstation. The images were evaluated by the same radiologist (C.A.G.), who had 3 years of experience in the field of cardiovascular imaging and was blinded to the subject group, patient age and gender. End-diastolic and end-systolic endocardial and epicardial contours were semi-automatically traced. The papillary muscles were included as the part of the LV myocardial mass. Using the delineated contours, the left ventricular end-diastolic volume (LV-EDV) and left ventricular end-diastolic mass (LV-EDM) were calculated, and the data were normalized to body surface area (BSA). In addition, at the level of segments 8 and 9, the interventricular septal wall thickness (IVS-WT) was measured.

Pixelwise T1 relaxation time values were calculated using manual tracing to define the LV myocardial

circumference as the region of interest (ROI) on the mid-ventricular short-axis images (LV native T1 value). In addition, an ROI was placed on the interventricular septum (segments 8 and 9) using the same method (IVS native T1 value). Care was taken to avoid the subsumption of blood or other tissues, such as epicardial fat, which could affect the T1 values (Fig. 1).

In addition, pixelwise myocardial T2 map images were evaluated qualitatively and quantitatively with the same technique used to map the T1 ROI, and any subjects were exclude based on this assessment.

Statistical analysis

The data were expressed as the mean (standard deviation, SD) depending upon overall variable distributions. Normality was assessed using the Shapiro–Wilk test. The normally distributed data were analyzed by one-way ANOVA, followed by the Bonferroni post hoc test and an independent samples *t* test when appropriate. Qualitative data were analyzed with Pearson's Chi square and the Yates corrected Chi square test as appropriate. Correlations were estimated using Pearson's correlation coefficient. $P < 0.05$ values were considered statistically significant. IBM SPSS statistics version 23.0 for Windows was used for the statistical analyses.

Intraobserver intraclass correlation coefficients (ICC) were determined for quantitative analyses; the 95 % confidence intervals (CI) were also calculated. All T1 values were used with at least 3 weeks between analyses for the intraobserver agreement calculation of the quantitative analyses.

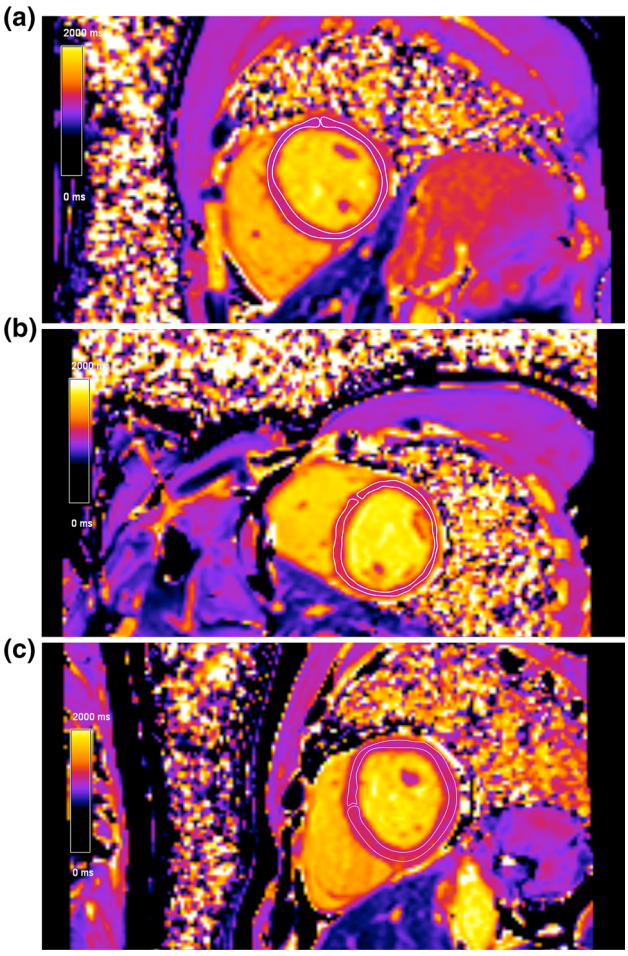

Fig. 1 Representative images of native T1 maps from **a** the non-athletic control group, **b** athletes who had <5 years of sports activity, and **c** athletes who had ≥5 years of sports activity

Results

Groups 1 and 2 had similar demographic data (gender, age); BSA; systolic blood pressure; and diastolic blood pressure, as shown in Table 1.

There were significant differences between groups 1 and 2 in the CMR imaging findings of the LV-EDV, LV-EDM, LV native T1 value, IVS-WT and IVS native T1 value ($P < 0.05$) (Table 2).

A study on the structural features of athlete's heart suggested that <5 years of training would not lead to any specific risk for cardiac disease in young athletes [18]. The athletic group was separated by duration of sports activity of <5 years (group 2a) and ≥5 years (group 2b) for further analysis. The demographic variables between groups 2a and 2b are shown in Table 3. All CMR imaging findings (LV-EDV, LV-EDM, LV native T1 value, IVS-WT and IVS native T1 value) were significantly higher in group 2b compared to groups 1 and 2a ($P < 0.05$). Although there were no significant differences ($P > 0.05$) in the LV-EDV, LV-EDM, and IVS-WT between groups 1 and 2a, significantly higher results ($P < 0.05$) were shown in the LV and

Table 1 Clinical and baseline characteristics

Parameter	Group 1	Group 2	*P* value
Sex (%)			
Male	26 (63.4)	28 (62.2)	
Female	15 (36.6)	17 (37.8)	ns
	Mean ± SD		
Age	24.4 ± 3.6	24.5 ± 3.05	ns
BSA (m^2)	1.89 ± 0.116	1.88 ± 0.107	ns
SBP (mmHg)	122.9 ± 4.7	123.1 ± 4.5	ns
DBP (mmHg)	78.7 ± 4.4	78.9 ± 3.7	ns

BSA body surface area, *SBP* systolic blood pressure, *DBP* diastolic blood pressure, *ns* nonsignificant

Table 2 CMR findings of group 1 and 2

Parameter	Mean ± SD		*P* value
	Group 1	Group 2	
LV-EDV(ml/m^2)	92.8 ± 6.9	105 ± 12.3	<0.001
LV-EDM (g/m^2)	41.7 ± 5.2	48.5 ± 6.7	<0.001
LV native T1 value (ms)	1174 ± 36.4	1230.5 ± 38.8	<0.001
IVS-WT (mm)	8.5 ± 0.8	9.2 ± 0.9	<0.001
IVS native T1 value (ms)	1179.8 ± 27.4	1267.7 ± 47.6	<0.001

LV-EDV left ventricular end-diastolic volume, *LV-EDM* left ventricular end-diastolic mass, *LV* left ventricular, *IVS-WT* interventricular septal wall thickness, *IVS* interventricular septum

Table 3 The demographic variables between group 2a and group 2b

Parameter	Mean ± SD		*P* value
	Group 2a	Group 2b	
Age (years)	22.3 ± 1.2	27.1 ± 2.3	<0.001
Years of sports activity (years)	2.8 ± 0.6	8 ± 1.6	<0.001
Endurance training (h/week)	9.5 ± 2.5	8.6 ± 2.5	0.2
BSA (m^2)	1.86 ± 0.115	1.9 ± 0.96	0.42
SBP (mm/Hg)	122.5 ± 4.9	123.8 ± 4	0.59
DBP (mm/Hg)	78.1 ± 3.8	79.6 ± 3.6	0.44

BSA body surface area, *SBP* systolic blood pressure, *DBP* diastolic blood pressure

IVS native T1 values in group 2a. These results are summarized in Table 4.

Moreover, although there was a moderately positive correlation between the LV native T1 and IVS native T1 values in group 1, a significant positive correlation was found in group 2a, and a superior positive correlation was found between those values in group 2b (Pearson correlation: 0.858, $P < 0.001$ for group 1; Pearson correlation: 0.947, $P < 0.001$ for group 2a; Pearson correlation: 0.979, $P < 0.001$ for group 2b).

Based on these results, a significant positive correlation was noted in athletes (groups 2a and 2b) between the LV-EDM and LV native T1 value (Pearson correlation: 0.919, $P < 0.001$ for group 2a; Pearson correlation: 0.953, $P < 0.001$ for group 2b) (Fig. 2) as well as a significant positive correlation between the IVS-WT and IVS native T1 value (Pearson correlation: 0.814, $P < 0.001$ for group 2a; Pearson correlation: 0.916, $P < 0.001$ for group 2b).

The ICC for the LV native T1 values was 0.98 (95 % CI 0.96–0.99), indicating nearly perfect agreement. The ICC for the IVS native T1 values was 0.95 (95 % CI 0.89–0.97), which also represents a very good agreement. Therefore, we can conclude that the output does not provide variance components.

Discussion

This blinded intraobserver study showed that all CMR imaging findings (LV-EDV, LV-EDM, LV native T1 value, IVS-WT and IVS native T1 value) were significantly higher in the athlete group than in the non-athletic control

Table 4 Comparison of CMR findings between subgroups

Parameter	Group 1	Group 2a	Group 2b
LV-EDV (ml/m^2)	92.8 ± 6.9[a]	95.2 ± 5.6[a]	116.2 ± 7.3[c]
LV-EDM (g/m^2)	41.7 ± 5.2[a]	43.8 ± 3.5[a]	53.8 ± 5.5[c]
LV native T1 value (ms)	1174 ± 36.4[b]	1204.4 ± 23.2[b]	1260.3 ± 30.7[b]
IVS-WT (mm)	8.5 ± 0.8[a]	8.8 ± 0.8[a]	9.7 ± 0.8[c]
IVS native T1 value (ms)	1179 ± 27.4[b]	1235.8 ± 26.6[b]	1304.2 ± 39.5[b]

LV-EDV left ventricular end-diastolic volume, *LV-EDM* left ventricular end-diastolic mass, *LV* left ventricular, *IVS-WT* interventricular septal wall thickness, *IVS* interventricular septum

[a] No significant difference between group 1 and 2a

[b] Significant difference between all groups (group 1, 2a and 2b)

[c] Significant difference between group 2b and group 1–2a

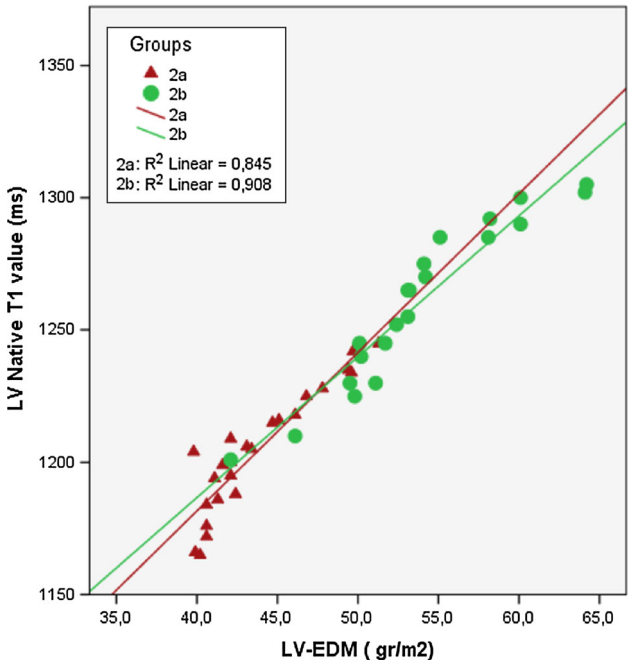

Fig. 2 Correlations between LV native T1 value and LV-EDM in groups 2a and 2b

group. We further demonstrated that although there were no significant differences in the LV-EDV, LV-EDM, and IVS-WT compared with non-athletic controls, the native T1 values were moderately higher in the athletes who had <5 years of sports activity.

CMR imaging offers higher precision for evaluating the LV-EDV and EDM due to its high spatial resolution [19–22]. This dependable, noninvasive imaging and analysis method for ventricular measurements is effective in identifying possible developing cardiomyopathies, which are the most frequent causes of SCD in athletes [22–26]. In this study, as in previous studies in the literature [22, 27, 28], we revealed that LV-EDV, LV-EDM, and IVS-WT are significantly higher in athletes than in non-athletic controls. Nevertheless, our results showed no significant difference between athletes who had <5 years of sports activity and non-athletic controls for these parameters.

In a recent review article, Doltra et al. [29] indicated that no study has examined an athlete's heart to determine an increased risk for SCD using the T1 mapping method. To our knowledge, this is the first study to evaluate myocardial fibrosis in athletes compared with a non-athletic control group with the T1 mapping technique using 3 T CMR imaging.

Athlete's heart, which is defined as the functional and structural adaptive changes in the heart in response to training, indicates a predisposition for myocardial fibrosis. Myocardial fibrosis, in turn, can lead to LV dysfunction, heart failure and SCD [30]. In this study, we did not use the LGE technique because the type of myocardial fibrosis in athlete's heart is 'diffuse myocardial fibrosis' (not focal) and because LGE cannot give a quantitative value for an objective assessment, as previously described [10]. Bull et al. [11] showed a clear correlation between native T1 values and histologic findings in diffuse myocardial fibrosis. Moreover, Hinojar et al. [31] demonstrated that abnormal native T1 values are compatible with ventricular remodeling. In addition, independent of the disease stage, the native T1 technique is a unique T1 mapping indicator that can distinguish between individuals at risk for SCD and healthy individuals. Previous studies have suggested that native T1 mapping can identify myocardial pathology before other CMR imaging techniques [31–34]. Based on this knowledge, our study revealed that native T1 mapping can detect myocardial changes at early stages of cardiac remodeling in athletes.

In this study, we demonstrated an increase in the native T1 values in the myocardia of athletes ($\sim$1230.5 $\pm$ 38.8 ms) compared to non-athletic controls ($\sim$1174 $\pm$ 36.4 ms) using 3 T MRI. This finding is a meaningful contribution to the literature and demonstrates that this imaging technique can be used to assess athletes' myocardial changes. In addition, native T1 values were significantly higher in athletes who had $\geq$5 years of sports activity ($\sim$1260.3 $\pm$ 30.7 ms) than in athletes of <5 years ($\sim$1240.4 $\pm$ 23.2 ms). However, in athletes who had <5 years of sports activity, a remarkable increase in T1 values was the most important result of the present study, but there were no considerable differences in the LV-EDV, LV-EDM and IVS-WT compared to non-athletic controls.

In addition, we focused on IVS native T1 values compared to LV native T1 values. This study revealed that in athletes who had $\geq$5 years of sports activity, there was a prominent rise in IVS T1 values compared to LV T1 values, with a significant positive correlation when compared to athletes who had <5 years of sports activity. That increase in the IVS-WT may affect the native T1 values more significantly than it would the remaining myocardium. Therefore, we suggest that evaluating the IVS with native T1 mapping can provide hints about cardiac remodeling.

Our study also showed a significant positive correlation in athletes (groups 2a and 2b) between the LV-EDM and LV native T1 value and between the IVS-WT and IVS native T1 value. We interpreted these findings as an indication that myocardial structural changes increase with the duration of sports activity, although this theory needs further investigation.

Several limitations should be considered in this study. First, it would be preferable to perform this study with a larger study population, although our sample size was determined by the power analysis to be sufficient. However, we were only able to include a limited number of

active sporting athletes. Therefore, the small size of our study population could limit the generalization of these findings. Second, because our study population did not have any cardiac complaints, we did not confirm our CMR imaging findings with histopathologic methods. Finally, the current evidence derived from the T1 mapping technique is considered less strong than similar evidence from the extracellular volume fraction method. To support our findings and define the underlying reason for the increase in T1 values in athletes, the use of this technique in athletes need further investigation.

In conclusion, this study explored whether the native T1 mapping technique has the potential to discriminate myocardial fibrotic changes in athletes when compared to demonstrated clinical or functional-morphologic imaging methods. Our study revealed that early myocardial alterations can be detected quantitatively by the native T1 mapping technique. The T1 mapping method might be a feasible technique to evaluate athletes, as it does not involve contrast, is non-invasive and allows for easy evaluation of myocardial remodeling. Further investigations are needed to define the native T1 cut-off values for risk classifications in athletes.

Compliance with ethical standards

Conflict of interest The authors declare no potential conflicts of interest with respect to the research, authorship, or publication of this article. The authors state that they have full control of all primary data and that they agree to allow the journal to review their data if requested.

Informed consent Informed consent was obtained from each patient prior to the examination in accordance with local Ethics Committee requirements.

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